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Jason Schoenfeld, MSIII
Thomas Jefferson University

Efstathia Andrikopoulou, MD
Thomas Jefferson University

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A CASE OF A 32-YEAR-OLD FEMALE WITH RIGHT-SIDED FACIAL DROOP AND NUMBNESS

Jason Schoenfeld, MSIII and Efsthia Andrikopoulou, MD

Case Presentation

A 32-year-old Hispanic female with a past medical history of asthma, depression and insomnia presented to the emergency room with complaints of right-sided facial droop associated with ipsilateral facial numbness and diminished taste for the past three days. In addition, she reported a moderate to severe pulsatile headache for the past day. This headache was initially right-sided and frontal, but then became right-sided and occipital in location. She had a history of a similar headache episode four years ago. Additionally, she noted moderately severe intermittent chest pain described as sharp then dull occurring over the past day. On review of systems, the patient had similar symptoms of facial droop and numbness without alteration in taste, one month prior. At that time, she was evaluated at an outside hospital and her symptoms had been attributed to a quetiapine-induced acute dystonic reaction which was treated with diphenhydramine with resolution of symptoms. Additionally, the patient denied sexually transmitted diseases, travel or outdoor exposure to tick-borne illnesses.

Physical examination revealed right-sided facial droop involving the corner of the mouth, with sparing of the forehead. She had decreased sensation to light touch on the entire right side of her face and preservation of sensation on the opposite side. All other cranial nerves were intact. The patient's cardiovascular exam was normal. At this point, the differential diagnosis for the patient included cerebrovascular accident, intracranial mass/lesion (tumor, aneurysm, arteriovenous malformation (AVM)) compressing cranial nerves V, VII and XII, manifesting itself as polycranial neuropathy, atypical Bell's palsy, Lyme disease, atypical episode of migraine headache or neurosyphilis.

Initial laboratory work-up including complete blood count, electrolytes, hepatic function panel, lyme antibody, and rapid plasma regain (RPR) was unremarkable. Urinalysis and urine culture were negative. All imaging including chest radiograph, computed tomography (CT) scan without contrast of the head and carotid ultrasound was unrevealing. Lastly, a magnetic resonance imaging (MRI) of the brain was performed showing non-specific changes of the bone marrow, which could correlate with anemia or anemia-like states.

Based on the results of her work-up, the patient was diagnosed with idiopathic unilateral facial nerve paralysis, Bell's palsy. We will review the work-up and characteristics of idiopathic peripheral facial nerve, CN VII palsy, as well as detail the differential diagnosis of secondary peripheral facial nerve palsy.

Differential Diagnosis

Physical examination is essential to differentiate a peripheral facial nerve palsy from a central facial nerve palsy.¹ The

peripheral lower motor neuron palsy classically manifests as unilateral impairment of movement in the facial muscles, drooping of the brow and corner of the mouth, and impaired closure of the eye and mouth. These symptoms are ipsilateral to the side of the lesion.¹ This presentation is in direct contrast to a central facial palsy, in which an upper motor neuron deficit causes weakness of the lower face contralateral to the side of the lesion. The forehead can still be innervated due to both ipsilateral and contralateral central innervation of the forehead.¹

A diagnosis of true idiopathic peripheral facial palsy, Bell's palsy, is made after exclusion of all other possible etiologies.² The significance of identifying other causes of secondary peripheral facial nerve palsy allows specific therapeutic options targeting the underlying disease.² The most common causes of secondary peripheral facial nerve palsy along with their characteristic signs, symptoms, laboratory and imaging findings are displayed in Table 1.

Management

Eye care of patients with Bell's palsy focuses on protecting the cornea from abrasion and drying due to malfunctioning tear production and lid closure.¹ Patients are instructed to use lubricating drops and ointment, and to report new findings such as pain, discharge, or changes in vision to a physician.¹

Corticosteroids, including prednisolone, have historically been used in the acute phase of Bell's palsy treatment.¹ It has been postulated that inflammation and edema lead to paralysis of the facial nerve. Thus, anti-inflammatory medications are recommended.¹ Anti-viral medications, including acyclovir and valacyclovir, have also been used concurrently with prednisolone based on the aforementioned link between HSV and facial paralysis. Until recently there was no strong evidence to suggest the impact, if any, that these drugs have on the natural course of the disease.

A 2007 randomized, double-blinded, placebo-controlled trial conducted by Sullivan et al., recruited patients with Bell's palsy who presented within 72 hours after onset of symptoms. Patients were randomly assigned to receive 10 days of treatment with prednisolone, acyclovir, both agents, or placebo. They found that early treatment with prednisolone, 50 mg per day for 10 days, started within 72 hours of onset significantly improved chances of complete recovery at both 3 and 9 months. They did not find any evidence of a benefit of acyclovir given alone, or an added benefit of acyclovir with prednisolone.³ Other studies, such as Engstrom et al., found similar results, but used an initial dose of prednisolone 60 mg daily for 5 days followed by a rapid taper with a total treatment time of 10 days.⁴ Based on these studies, if a patient presents within 72 hours of onset, prednisolone should

Table 1. Causes of secondary peripheral facial nerve palsy and their respective key diagnostic physical, laboratory and imaging findings

Cause	History	Physical Exam	Laboratory	Imaging
Lyme disease	Tick bite	Skin rash (<i>erythema chronicum migrans</i>) – if it is present, it is pathognomonic Arthritis and radiculoneuropathy		IgG/IgM titers
Herpes Simplex Virus-1 (HSV-1)		Mucosal lesions of mouth, gingivae, pharynx, lips (recurrent herpes labialis) genitals. Herpetic whitlow, encephalitis, eye infections		
Herpes Zoster Virus		Erythematous vesicular rash on ear (<i>zoster oticus</i>) or in mouth		
Trauma	History of head trauma or intracranial surgery			
Tumors		Presence of more than one CN palsies, e.g. CN VIII palsy leading to hearing defects/loss		
Guillain-Barre Syndrome	Prior respiratory tract or gastrointestinal infection, most commonly <i>Campylobacter jejuni</i>	Ascending motor paralysis, diminished or absent muscle stretch reflexes, and sensory loss	Albuminocytologic dissociation on CSF analysis	
Neurosarcoidosis			Hypercalcemia, elevated serum ACE levels, elevated serum alkaline phosphatase and increased urinary calcium excretion	Chest radiography and Gallium-67 scan to reveal the presence of mediastinal/hilar enlargement due to lymphadenopathy and/or interstitial lung disease
HIV	Prodrome with fevers, night sweats, lymphadenopathy, weight loss and myalgias		HIV serology	

be initiated in either of the two described regimens. If a patient presents after 72 hours of onset, there is no current evidence to suggest that administering prednisolone has any benefit.

In a 2008 Cochrane review of seven randomized clinical control studies performed by Lockhart et al, there was no significant difference in the rate of incomplete recovery at one year of patients with Bell's palsy treated with antivirals as compared to placebo [RR 0.88 (95% CI 0.65 to 1.18)].⁵ Furthermore, the outcome in patients treated with antivirals was significantly worse than those treated with corticosteroids [RR 2.82 (95% CI 1.09 to 7.32)]. Additionally, there was no significant difference in long-term sequelae (i.e. crocodile tears or motor synkineses) in the antivirals and corticosteroids group versus the corticosteroid monotherapy group.⁵ Based on these results, antiviral

medications are not currently recommended as first-line therapy for the acute treatment of Bell's palsy.⁵

In addition to pharmacologic therapy, various forms of physical therapy, including exercise, mime, biofeedback, laser, massage, and electrotherapy, have historically been used in the management of acute Bell's palsy.⁶ While individualized studies have previously demonstrated the efficacy of these techniques,^{7,8} recent systematic reviews have called into question whether physical therapy offers any benefit in time to recovery or reduced long-term sequelae as compared to controls.^{9,10} It is apparent that further research with randomized trials are needed to better assess whether these techniques should be routinely offered to Bell's palsy patients.⁶

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"The Horror"

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